15

Smoking Depresses Adipose Lipoprotein Lipase Response to Oral Glucose

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Adipose tissue lipoprotein lipase was studied in smokers (n = 17) aged 18-47 years and compared to enzyme activity in non-smokers of comparable age (n = 8) and a second time in some subjects 5-9 weeks after cessation of smoking (n = 7). Serum cotinine levels served to validate the smoking status of the subjects. Fasting enzyme activity was similar in smokers and non-smokers, when expressed per 10^6 cells, but was significantly increased when normalized for cell size. When lipoprotein lipase was determined in the same individual 4 hours after an oral glucose load, a significant decrease ($p < 0.002$) occurred in the smokers, while enzyme activity rose in the non-smokers ($p < 0.02$). A tendency for enzyme activity to rise after oral glucose was seen in ex-smokers, which did not reach statistical significance. Even though the mean serum insulin and glucose levels did not differ in the 3 groups of subjects, the % decrease in lipoprotein lipase after oral glucose in smokers was negatively correlated with insulin release into serum in the same subject, i.e., the greater the insulin release, the less the decrease in lipoprotein lipase activity. We would like to propose that the lower body weight in smokers is related to the paradoxical response of adipose tissue lipoprotein lipase to carbohydrate and that the reversal of this behavior contributes to the weight gain often observed after cessation of smoking.